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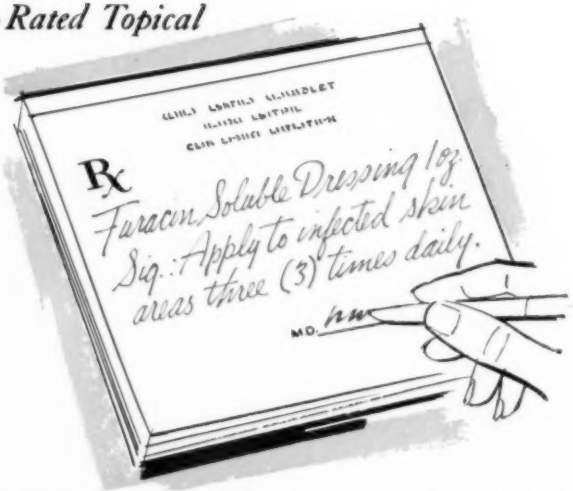

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CONTENTS

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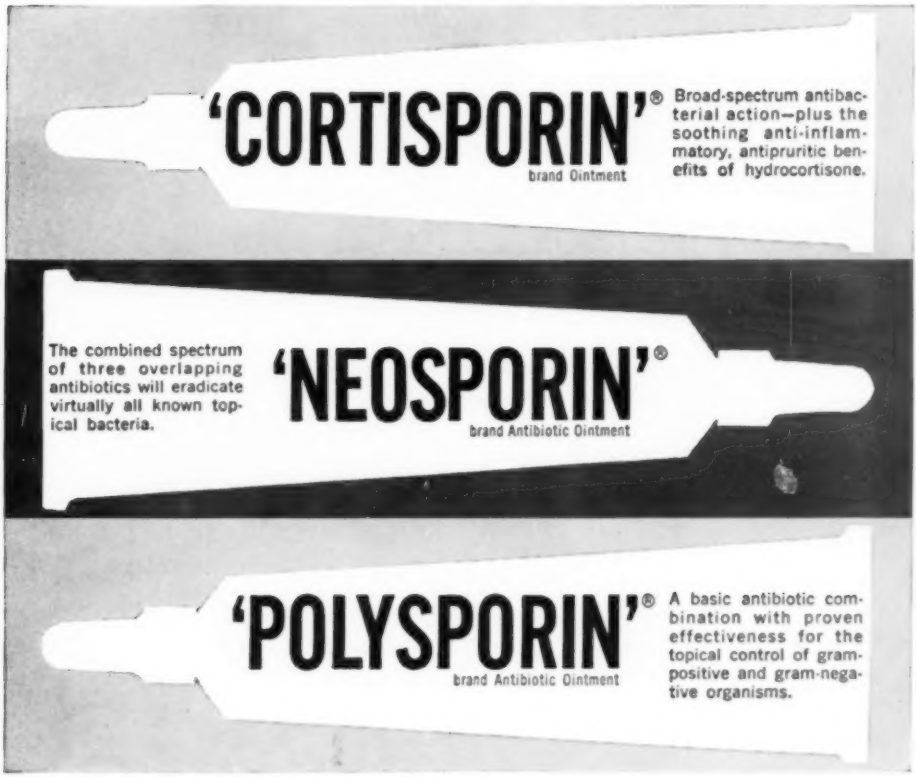

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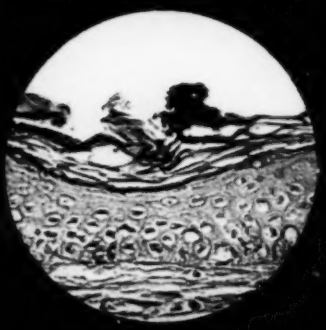

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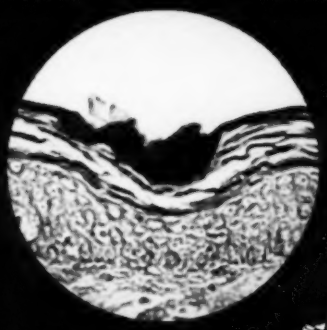

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Agnathia Associated with Pharyngeal Isthmus Atresia and Hydramnios

Warren W. Johnson, M.D.*
John B. Cook III, M.D.*
Tennessee

Since the description by Treacher Collins in 1900 of the clinical manifestations of mandibulofacial dysostosis, numerous variations of malformation of the first pharyngeal arch have been observed. These include microagnathia with ptosis of the tongue, described by Pierre Robin in 1923,[1] cheiloschisis and cleft palate, malformation of the external and middle ears, microagnathia (or mandibular hypoplasia),[2] hypoplasia of the mandible with cleft palate, as described by Callister in 1957,[3] bifid tongue,[4] hypertelorism,[5] congenital deaf-mutism, unilateral underdevelopment of the mandible (also reported in sheep),[6] as well as slightly abnormal development of any portion of the first pharyngeal arch derivatives.[7]

The normal embryology[8] and adult derivatives[9,10,11] have been clearly described, and with the results of experimental embryology by Speeman,[12] Stockard,[13] and others, anomalies of the first arch became accepted as 'arrests in development'. While the genetic implications were recognized,[14] the degree of penetrance was first considered to be dependent upon, or secondary to, vascular developmental anomalies of the region by McKenzie in 1958.[7]

Although various types of first pharyngeal arch malformations of a significant degree are said to be fairly rare, the total number has been estimated as "not inconsiderable".[15] The extreme degree of malformation in this infant is of interest, inasmuch as complete absence of the mandible is accepted by most authors as being rare. Also of interest is the associated hydramnios which has been associated with malformations, especially of the central nervous system (and most frequently with anencephalus).[16,17]

Clinical Data: The mother, a 27-year-old negro, gravida one, having been examined once in the out-patient clinic, came to the John Gaston Hospital of the City of Memphis Hospitals after the

* Institute of Pathology and Bacteriology, University of Tennessee School of Medicine

onset of labor at 36 weeks gestation. Other than hydramnios, (8,000 cc.), there were no complications of delivery. At the time of delivery it was found that the infant had a malformation of the lower face with only a tiny oral orifice. An attempt to insert a rubber catheter through the tiny oral orifice of the infant allowed only a few centimeters of catheter to pass with no material aspirated. A tracheostomy was performed, but vital signs ceased after 20 minutes.

Placenta: This structure was markedly flattened (fig. 1), measuring 22.5 x 16.5 x 1.5 centimeters, and weighing 450 grams. A small, marginal infarct, old, was present, and the cord was inserted 1 centimeter from the margin. The membranes were complete and the maternal surface was intact. Microscopic examination revealed slight prematurity as evidenced by distinctly less than 30% of the chorionic villi possessing synctial clumping ("knots"), and the fetal vessels being near the villus margins. The placenta was, therefore, slightly premature histologically, but weighing as much as a full-term placenta.[18]

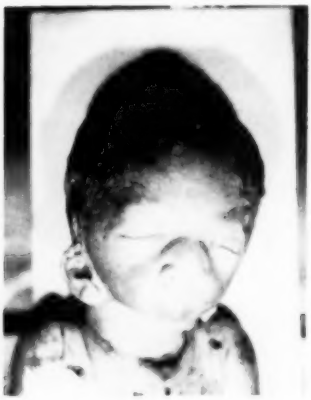

FIG. 1

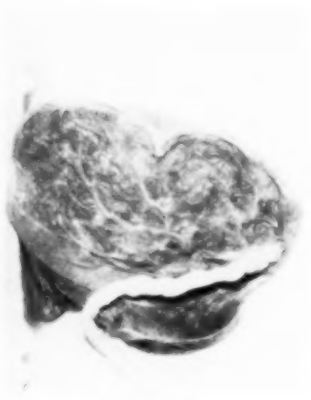

FIG. 2

Necropsy Findings: The infant was slightly premature, measuring 47 centimeters crown-heel length, and weighing 1910 grams. The ears were placed very low on the neck (figs. 2, 3, 4), and the oral orifice was extremely small, the greatest diameter being 10 millimeters. The malar (zygomatic) bones were flattened, with no zygomatic processes present. The mandible was completely absent, as were all muscles of mastication. The maxilla was markedly reduced

in size containing anlagen for 20 teeth. There was a bifid structure, containing loose fibrous tissue and a moderate number of capillaries, which arose in the posterior blind oral pharynx. The posterior two-thirds of the palate was cleft, (fig. 5). The tongue consisted of an oval elevation immediately above the larynx (fig. 6), with a small tube-like opening extending from the larynx to the small choanae.

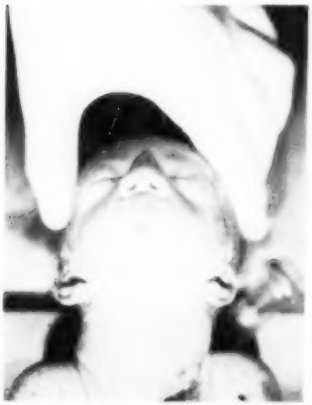

FIG. 3

FIG. 4

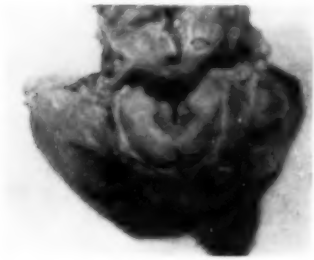

FIG. 5

FIG. 6

The hyoid bone was present; however, many branches of the external carotid arteries were either absent or diminutive. The posterior auricular and occipital branches were well defined. The superior thyroid branches were slightly larger than normal, and there was a large branch on the right side, corresponding to the right lingual artery, which terminated in the connective tissue of the

floor of the mouth and in the anomalous bifid structure at the blind end of the pharynx. A large anastomosis between the external carotid arteries was present immediately behind the hyoid bone (fig. 7).

Other than the low position of the auricles, and short external meatal canals, no anomalies of the ears, including the middle ears, were present. All 3 ossicles were present, and without gross or microscopic malformations, bilaterally.

FIG. 7

The buccal fat pads were large, as were the sub-maxillary glands. The left lobe of the thyroid gland was present; however, the right lobe was absent. The thymus gland was large, weighing 20 grams, with the expected weight being approximately 9 grams. The adrenals and pituitary gland revealed no significant changes. There were multiple interstitial pulmonary hemorrhages and atelectasis, and unruptured sub-capsular hepatic hemorrhages.

DISCUSSION

The relationship of congenital anomalies to the amount of amniotic fluid, in many but not all instances, has been placed in three categories:[19] those with a normal amount of amnionic fluid, e. g., hydrocephalus; those with a reduced amount of amnionic fluid, e. g., incomplete urethra, (however, this is inconsistent, and hydramnios has been reported as accompanying such genito-urinary malformations); and thirdly, those with increased amnionic fluid, e. g., anencephaly and esophageal atresia. In this infant, the complete atresia of the oral pharynx, as well as the severe microstomia

would simulate esophageal atresia insofar as the inability of the fetus to swallow amnionic fluid is concerned. It is presumed that in anencephalus the severe central nervous system malformation renders the swallowing mechanism incompetent on a neurogenic basis.

The placenta in fetal malformations is said to be large in those instances associated with hydramnios.[16] In this instance, the placenta weighed that of the average term placenta; yet, was slightly premature by history as well as microscopically. Hence, it may be considered slightly larger than normal.

The genetic aspect of first pharyngeal arch malformations has been studied,[14] and appears to be a non-sex linked, dominant gene, which may 'skip' a generation, with the severity (penetrance) dependent upon the degree of associated vascular aberrations.[7] Although somatic (or mesodermal) anomalies undoubtedly can result from vascular malformations, the fact that the vessels revealing malformations in this infant are so closely associated with the tissues within which the vessels develop, it would appear difficult to conclude, definitely, that the gross anomalies are strictly secondary to the vascular malformations, rather than concomitant abnormal development of both. The latter possibility would appear more likely in pharyngeal arch anomalies than in other locations. Certainly not all deformities have been demonstrated as secondary to vascular aberrations, e.g., branchal pouch cysts and malformations of the external ears.

Maternal malnutrition in rats has been shown to result in malformation of the fetus.[20] However, as was demonstrated by Stockard,[13] and other experimental embryologists, the time of insult to the developing embryo, more than the nature of the injury, is of greater importance, so that during the fourth and fifth weeks, and perhaps the sixth, the injury to the facial anlage is of the greatest significance.

In 1957 Warkany pointed out the value of post mortem examination in helping to establish a more complete picture in each instance of death due to congenital malformation.[21]

Absence of malformation of middle ear derivatives of the first pharyngeal arch (the malleous and incus) in severe microagnathia has led some authors to question the derivation of these structures from Meckel's cartilage.[6] However, embryologists have always considered these to be first arch derivatives,[8] and we have traced,

in serial sections, Meckel's cartilage in a 55 millimeter crown-rump human embryo (approximately 12 weeks gestation) directly into the malleus cartilage. It does not appear unreasonable that a portion of the cartilage bar of the first pharyngeal arch should be involved in malformation with another portion remaining normal in some instances, any more than that one side of the arch should be defective while the opposite side remains uninvolved, as has been reported in man and in sheep.[6]

Smith and Stowe[22] reviewed 39 cases of Pierre Robin syndrome in which one-fourth of the cases had a history of an intra-uterine disturbance in early pregnancy. Of the 15 patients undergoing ophthalmological examination, 9 major eye lesions were found. Fourteen of the infants weighed less than 2,500 grams. No instance of hydramnios was described in their series of cases. The association between recurrent otitis media and cleft palate was reiterated by the authors. Major otological abnormalities were present in 3 infants and minor defects in 8 others. One abnormality consisted of the absence of the left middle ear.

SUMMARY

A premature infant with complete absence of the mandible and atresis of the pharyngeal isthmus, with associated vascular anomalies has been described in a case of hydramnios.

A review of the development of the concept of first pharyngeal arch malformations has been given with mention of the hereditary predisposition and possible importance of maternal gestational factors to such lesions.

Questions by previous authors as to the contribution to middle ear structures by the first pharyngeal arch have been considered.

The association of hydramnios with congenital anomalies has been observed with special emphasis upon certain types of malformations.

CONCLUSIONS

The same mechanism causing hydramnios frequently observed in instances of anencephaly and esophageal atresia has been proposed as operative in this infant.

The absence of middle ear involvement in this infant with agnathia indicates that portions of the visceral mesoderm of the first pharyn-

geal arch (Meckel's cartilage) can be malformed without total involvement.

The suggestion is made that the vascular malformations in this infant occurred concomitantly with the first pharyngeal arch anomalies, rather than being the cause of the latter.

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851 *Madison Ave., Memphis* 3

Children at a Health Museum

BRUNO GEBHARD, M.D.*
Ohio

PHYSICIANS' attitudes of how much and what the patient should know about his body and his ills have changed considerably in the last twenty years, from "they don't know anything" to "they know too much and are over-anxious about their health."

In our age of mass communication the ratio of sound health information to unsound misinformation spread via newspapers, magazines, even best sellers of books, radio and television, is at its best a fifty-fifty proposition. Competing for the people's dollars to the so-called "Health" drives spreads more disease information than factual health facts. To remedy this situation—at least on the local level—a new community health facility was incorporated as the first one of its kind in the U. S. A. in 1936 and opened to the public in 1940 under the name "Cleveland Health Museum." This museum is an outgrowth of the health education activities of the Cleveland Academy of Medicine. Its charter states as the main purpose, "to educate the public in matters of health" and "to operate and maintain a museum of health and hygiene."

Health museums have as their forerunner the hygiene museums of Europe, especially the German Hygiene Museum in Dresden and the health expositions of Europe. The Century of Progress Medical Exhibits (Chicago, 1933-34) and those of the New York World's Fair (1939-40) and locally, the Great Lakeside Exposition (1936-37) provided the stimulus for the establishment of a permanent facility of planned, all-year-round activities in community health education, with stress on the use of visual means as exhibits, motion pictures, and more recently, television.

The philosophy of a health museum is based on the premise that diseases are doctors' business, but health is everybody's concern. Today's patients are no longer content in just taking as

* Director, Cleveland Health Museum

Wonder of New Life Room — General View

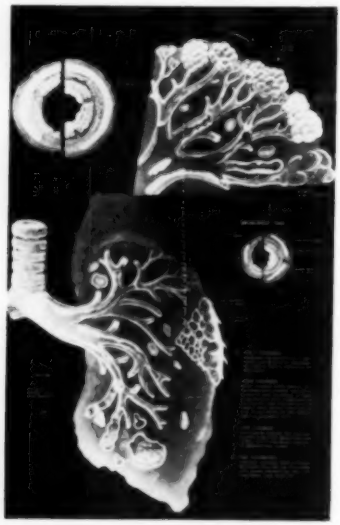
an answer to their questions, the standard phrase "doctor's orders." He, or she, and this includes children, wants to know "why?" The public is no longer just on the receiving end of medical care. The idea that diseases can—and should be prevented—has taken a hold on the public to a much larger degree than most physicians realize. How real and to what an extent there is a "crisis of American medicine" is not in the scope of this paper to answer, but physicians will do well to take a more active part in the leadership of all forms of health education at their offices, at the bedside, or on the lecture platforms.

The general practitioner has more often a chance to apply preventive medicine in pediatrics and obstetrical cases than in any other field. Full cooperation of the patient and his or her family calls for mutual respect between physician and patient and the willingness to do right. The last one usually prevails with parents being concerned for their children. People who might not care much for their own health want to do right by their offsprings. This means that we must have patients who are not biologically illiterate, who know the fundamentals of health care and home nursing, and are willing and able to apply such knowledge, and

last, but not least, must have the means to do so, either by themselves or through community facilities.

Health museums are not watered-down medical museums. They have their place in professional education. As all hygiene is nothing more than applied biology, health museums feature normal growth and development of the human being from "egg to eighty" with stress on family life education and man-made diseases. We try to avoid the over-specialization in medicine and dentistry and appeal to the self interest of the normal individual.

A health museum is a place where people can inform themselves about themselves. It is a science museum specializing in personal and public health. At a museum one can see what up until now one has only heard or read about. Curiosity is not only of the morbid kind but very natural with everybody and especially with growing children. Besides the very young ones, there are other age groups



Lung With Cystic Fibrosis

where interest in health is great, as the expectant parents, or the middle-aged man or woman who get worried about their weight or their heart. Physicians who are afraid that health education leads to unwarranted self diagnosis should never forget that it is always the patient who makes the first "diagnosis." If he or she

do not realize that something is wrong with their health and that professional attention is necessary, they would never come to see physicians in the first place, and this happens more often than most M.Ds. realize.

Exhibits are the backbone of any museum, and activities of all kinds put life into them, so do especially our nearly 30,000 young visitors during a year's time. Our exhibits are of two kinds—permanent ones and special exhibits. Permanent ones deal with human biology. The best known of these is Juno, the transparent woman's figure, and especially our collection on the Wonder of New Life dealing with human reproduction (illus. 1). Special exhibits of from one to two months' duration deal with all kinds of subjects. Some are geared to the seasons like "Hay Fever", in connection with which we have publicized via newspapers and radio, a daily ragweed pollen count for the last twenty years. Exhibits on Polio stress the need for "shots." A special exhibit appealing especially to children was the one on "Who's Zoo—Animals as Pets or Pests", including a section on "Animals in Medical Research" which did not even bring us a protest from our local Anti-Vivisection Society. The special exhibit on "Fun in the Sun and At The Sea" gave advice on first aid under camping conditions, how to avoid sunburn and accidents in skin diving and was planned with the help of the local Red Cross Chapter. "Man in Space", which went on display three months after the first Sputnik, was our most successful one, attendance-wise, dealing with the physiology of space travel. A student group from a Catholic high school established a tracing station right on the roof of our Museum.

Under our permanent exhibitions the most outstanding one is the "Wonder of New Life". The Museum is the proud owner of nearly one hundred sculptured, life-sized models by the late Robert L. Dickinson, M.D., dealing with all phases of human reproduction from fertilization through the growth of the embryo, the actual process of delivery, and of other items as fetal circulation and multiple births. This collection includes also standard models of the average American young man (Norman) and his partner (Norma). These sculptures are based on 35,000 measurements. The physiology of male and female reproductive organs are demonstrated in several animated displays. Basic principles of heredity and genetics are shown in push-button exhibits as "Father Determines Sex." The "Hormone Lady" gets great atten-

tion by young and old. "Life's Beginnings" is the title of an illuminated, six-foot in diameter, plastic model of an egg in the process of fertilization.

"Where do you get your exhibits?" we are often asked. Most of them are our own production. Up to one-half dozen museum's artists and craftsmen with additional help from Junior League volunteers produce not only displays for ourselves but also make duplicates on a cost basis available to other museums, colleges and schools. On special request we construct professional exhibits as "Accidental Poisoning in Children", "Saving Face (Orthodontics)", and "Lung with Cystic Fibrosis". We offer a consultant's service to physicians and health agencies to help them with their exhibit problems. A limited number of exhibits are available on a loan basis for the increasing number of "Health Fairs" around the country.

Admitting that too many museums have the atmosphere of mausoleums, one has to see for himself that this is not true of a health museum[1]. Health museums are not a memento mori—as hospitals are still for many patients—but a memento vitae. We might be accused of being biased but in our twenty years of operation we already have the second generation of enthusiastic visitors coming to us. These are the young families with their small children whose Mom and Dad first came to us as high school students. These young families constitute a growing part of our dues-paying ($10 minimum) membership of nearly six thousand individuals and corporations.

Museums are basically places for informal education of all age groups, but since 1951, when our department of education was formally established, demands for class instruction have more than tripled. Besides our own staff of three health educators, one full-time science teacher is assigned to us by the Cleveland Public School system, another part-time teacher comes from a suburban school system. Other boards of education reimburse us for our school education services on a contract basis. The last ones are mostly smaller systems which have neither the necessary visual aid material nor enough well qualified teachers in the biological sciences and health education.

The Museum reaches out into the community in many ways. Staff members, for example, serve as resource persons to the Pre-Med Club of the Shaker Heights Senior High School which

has a membership of more than one hundred students. Their current study project is on blood and blood groups.

One of the most urgent subjects in health education is sex education which since 1940 has been one of our prime activities. Sex seems to have troubled mankind since the days of Adam and Eve. It is, therefore, natural that it troubles just as much today's parents, teachers and even occasionally physicians. There is more to sex education than just knowing the so-called "facts of life" which are so often fictitious, but to know the right thing at the proper time is an imporant first step. We have, therefore, organized class instructions on "menstruation" mainly for fifth graders. It is always the teacher who decides when and under what conditions (boys and girls together, girls only) such instruction takes place. Visits by Girl Scout Troops are regular affairs on Saturdays and as their mothers bring them in their cars they also participate and often learn too. We wish we would have more "Father and Son" activities along those lines. Just to keep the record straight, parochial schools make use of our facilities just as well as public and private schools. As good discussion is essential to learning and to attitude formation, especially in this hush-hush field, ample time and opportunities are given for this purpose by our instructors. Showing a 15-minute film usually breaks the ice for this discussion and the highlight is a visit—the word comes from vision—to the Wonder of New Life Room which provides for a unique experience with the minimum of emotional trauma, in an atmosphere conductive to maximum learning and retention of facts, which the classroom just does not offer.

When the time allows children have a "browsing period" where they can make their own discoveries in whatever field they are personally interested. The Museum has neither uniform guards nor "do not touch" signs. Willful damage does not amount to fifty dollars a year with an attendance of nearly 80,000 persons. Parents are indirectly involved in this instruction as their permission is needed for field trips. Besides that many children unfortunately have to pay for bus transportation.

Class instruction is not limited to sex education—other subjects in great demand are the Five Senses, Nutrition, Dental Exhibits, Personal Health, and Community Health. An illustrated brochure, available free on request for teachers and physicians, titled "Field Trips with a Difference"[2] describes these activities in detail. In

1959 nearly 20,000 school children, comprising 368 classes, received such instructions. In addition, 217 classes from schools, colleges, and schools of nursing toured the Museum. The 1960 figures will be nearly ten percent higher.[3]

Special attention is given to the exceptionally bright student, as well as to slow learners and those handicapped in hearing or vision.

New teachers of many school systems meet on Saturday morning to familiarize themselves with progress in health education. School nurses and dental hygienists have regular meetings at the Museum.

Evaluation of any education program is a difficult affair. It demands first proper record-keeping. What the patient's case history is for the physician are the "Group Instruction Reports" of our staff, written up in detail after each activity. They are the basis for visitor reaction studies. We record new questions brought up for discussion besides the many standard ones which each new generation will again bring up. Here are some questions from an 8th grade class: Does the cord really have to be tied and cut? Why are some babies born left handed? How much blood does a woman lose when she menstruates? How is milk produced in a woman's breast? How can a woman prevent the birth of a baby after she is two months pregnant? Why does the man's penis become hard when he holds a woman close to him? Can you give me a simple word for hormones? All questions asked are answered specifically by our instructors. What would be your answer to these questions asked by your own son, or a boy in your office?

In order to stimulate a follow-up of a museum's visit we issue "Attendance Certificates" to those children who work on a project. A recent group of 5th graders picked these subjects: Lymph Glands, Heart, Veins, Bones, Transparent Woman, Beginning of Life, Pituitary Gland, Kidneys. In an accompanying letter one girl wrote, "I like to listen to my voice on the telephone and test my eyes and also my strength. When it was time to go I was sad because I didn't want to go."

Another kind of follow-up are the week-end visits of families brought to the Museum by their children proudly showing off these exhibits which especially interested them, as the question and answer game on "How Many Calories?" or "How to Brush Your Teeth."

Volunteers from high schools help us during the summer months as junior guides, or in the workshops making exhibits.

The influence of a museum goes beyond its own walls. When the fight for fluoridation of drinking water was on in Cleveland (and later successfully completed) innumerable discussions in school rooms were held for which we provided color slides and charts. We are currently involved with another health agency to get a better understanding of epileptic children and to build a special exhibit on this subject.

Physicians are interested in our "quicky visitors"[4] who are not coming to see the Museum per se, but just coming to see what the wrist looks like which little Johnny broke, or his father might want to know what really slipped in the slipped disc.

The future of health museums in this country will depend very much on whether organized medicine, or more likely, individual physicians can see the advantages of this new type of health education. Dallas, Texas, opened a museum in 1946. The Lankenau Hospital in Ardmore, Philadelphia, pioneered one in 1953 as part of its hospital activities. In Hinsdale, Illinois, the Kettering Family Foundation in 1958 was the sponsor of a health museum which is rapidly growing and is located under the same roof as a medical arts building. Health galleries as part of a state museum exists on the campus of the Nebraska State Museum in Lincoln. Many science museums like Chicago, Boston and Buffalo have special sections on medical and health exhibits. Vivant sequentes!

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8911 *Euclid Avenue, Cleveland 6*

Activities of The Poison Control Center . . .

THE EPIDEMIOLOGY OF ACCIDENTAL CHEMICAL POISONINGS

HAROLD JACOBZINER, M.D.*
HARRY W. RAYBIN, M.S.**
New York

SINCE the Poison Control Center was established (March 9, 1955) through December 31, 1959, 33,883 poisonings were reported to the Poison Control Center for which aid relating to toxic ingredients and treatment was requested. The number of products involved exceeded a thousand and many of the products included 10 or more separate brands. For the sake of simplicity, these products have been grouped into large categories as shown in Chart I. It is seen that at all ages barbiturates are the chief offenders and aspirin follows closely behind as the second leading cause of all poisonings at all ages. Internal and external medications are responsible for nearly 58% of all poisonings. The bleaches, insecticides, rodenticides and lead also constitute great hazards.

Table 1 depicts poisonings by type of poison as well as by age. Nearly 45% of all poisonings at all ages occurred in children under 5 years of age, though they constitute only 8.3% of the New York City population. It is also apparent that 58% of all poisonings occurred in individuals under 20 years of age.

*Assistant Commissioner for Maternal and Child Health, Department of Health, City of New York
**Technical Director, Poison Control Center

CHART I — POISON CONTROL CENTER

NEW YORK CITY DEPARTMENT OF HEALTH
19,357 CASES OF POISONINGS REPORTED IN
CHILDREN UNDER 20 YEARS OF AGE
BY TYPE OF POISON - (1955-1959)

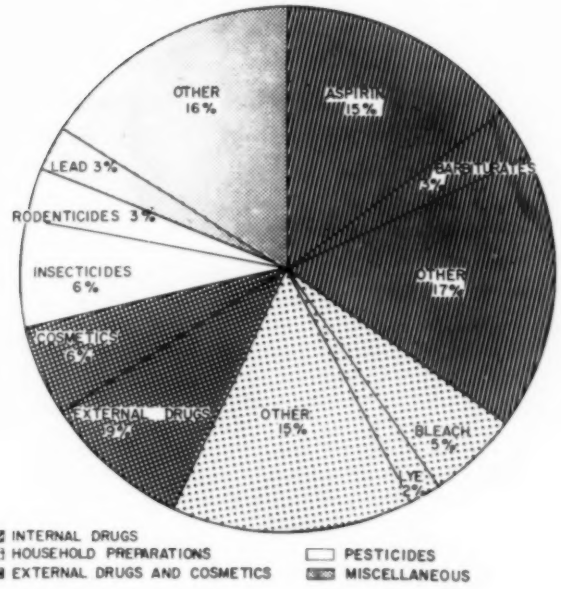


Chart II depicts poisonings in children under 20 years of age by type of poison on a broad percentage basis. One observes that certain changes in the proportion and percentage of poisonings by type of poison have occurred as compared with the chart showing poisonings at all ages. In individuals under 20 years of age, aspirin is the leading cause of poisonings, accounting for 15% of all incidents in this age group, and barbiturates, which were the chief offenders in the "all ages" category, now are relegated to a place of secondary importance, being responsible for only 3% of poisonings in this age group. This is undoubtedly due to the fact that barbiturates are used with great frequency in the older age group, not only as medications but also in intentional suicide attempts. The insecticides and rodenticides cause many poisonings in the younger age group and it is undoubtedly a function of growth and

TABLE 1

POISON CONTROL CENTER
NEW YORK CITY DEPARTMENT OF HEALTH
POISONINGS BY AGE
1955 - 1959

Type of Poison	Total	Under 1 Year	1	2	3	4	5-9	10-14	15-19	20 and Over	Age Unknown: Child	Age Unknown: Adult	Age Unknown: Not Spec.
Internal Medicines	16,513	159	883	2,242	1,419	500	309	225	864	9,244	74	270	524
Aspirin	5,493	38	240	1,141	798	240	82	64	251	545	26	6	46
Barbiturates	5,146	14	54	115	58	33	28	41	148	4,595	2	164	96
Other	7,874	107	581	986	563	219	199	120	465	4,506	46	100	182
External Medicines	3,041	135	496	470	206	93	149	59	189	1,125	21	41	61
Cosmetics	1,327	88	350	429	142	49	54	8	29	110	13	19	28
Household Prod.	4,560	170	1,299	963	330	160	153	55	181	1,020	70	60	99
Bleaches	1,314	54	308	265	99	51	43	25	110	336	9	14	20
Lye	466	11	88	85	68	40	27	8	17	101	12	5	8
Other	2,779	125	903	613	163	69	83	22	54	583	49	43	71
Solvents	1,327	45	437	292	88	36	43	18	64	257	12	23	27
Turpentine	277	10	104	69	16	5	12	2	14	37	2	1	5
Kerosene	210	10	93	55	13	7	5	2	5	14	2	1	5
Other	870	25	240	175	59	22	26	14	47	186	8	21	17
Insecticides	1,572	106	447	340	121	75	70	9	65	258	24	39	37
Rodenticides	678	37	164	122	67	28	23	7	28	172	4	4	22
Miscellaneous	4,035	260	1,192	1,119	404	223	110	101	46	725	82	187	136
Lead	682	7	149	273	120	40	35	5	1	24	2	1	5
Other	4,153	253	1,030	846	542	183	275	98	81	701	87	206	131
TOTAL	33,085	1,001	5,284	5,992	2,857	1,152	1,091	482	1,498	12,897	307	543	779
Percent	100.0	3.0	15.6	17.7	8.4	3.4	3.2	1.4	4.4	38.1	0.9	1.6	2.3

The figure 682 (lead) includes provisional lead poisoning cases

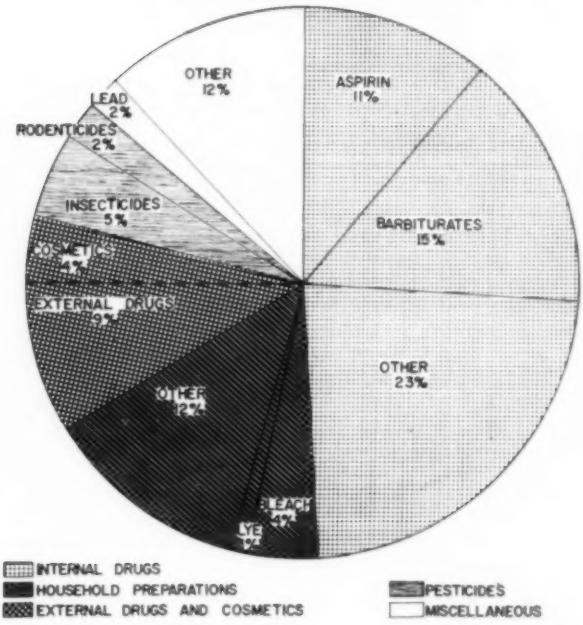
development and availability and accessibility, as will be discussed in greater detail subsequently.

Table 2 shows the incidence of poisonings by type of poison in children under 20 years of age on a more detailed numerical and age basis. It is again seen that aspirin is the number one offender and that internal and external medications are responsible for over 43% of poisonings in this age group. It is of interest that the type of poison varies with age and that in the "creeper" and "toddler", that is, in children under 2 years of age, poisonings result largely from products which are obtainable in open places and at low levels, such as under the sink, on the floor, or on low shelves or dressers, and that the major offenders are household products—solvents, rodenticides, insecticides and lead. As the child grows older—3 years and above—and becomes more mobile and able to climb to higher places (the "climber"), he is more apt to become involved with poisonings which result from ingestion of medications which he usually obtains either from the medicine cabinet or on a shelf.

Of the poisonings reported in children under 20 years of age, 78.2% occurred in children under 4 years of age and nearly 85% in children under 5 years of age. Age 2 years was the most dangerous age, with age 1 following closely behind. Together, these two

CHART II — POISON CONTROL CENTER

NEW YORK CITY DEPARTMENT OF HEALTH
33883 CASES OF POISONINGS REPORTED BY
TYPE OF POISON - (1955 - 1959)



ages were responsible for 58.2% of all poisonings in individuals under 20 years of age. This clearly shows that children under 5 years of age are a highly susceptible and vulnerable group.

WHO ARE THE CHIEF OFFENDERS?

Though the number of products involved may be legion, five substances were responsible for 30% of all poisonings in children under 20 years of age (Chart III). This is a highly significant finding since it clearly illustrates that the total prevention of poisoning is within the realm of possibility if only elementary precautionary measures are employed with regard to handling and, particularly, to safe storage of chemical products. We have shown previously that there is a distinct correlation between the occurrence of a poisoning and availability and accessibility of a product.

TABLE 2

POISON CONTROL CENTER
NEW YORK CITY DEPARTMENT OF HEALTH
POISONINGS REPORTED IN CHILDREN UNDER 20 YEARS OF AGE
1955 - 1959

Type of Poison	Total	Under 1 Year	1	2	3	4	5-9	10-14	15-19	Per Cent
Internal Medicines	6,601	159	883	2,242	1,419	500	309	225	864	34.1
Aspirin	2,870	38	248	1,141	798	248	82	64	251	14.8
Barbiturates	491	14	54	115	58	55	28	41	148	2.5
Other	3,240	107	581	986	563	219	199	120	465	16.7
External Medicines	1,795	135	496	478	206	81	149	59	189	9.3
Cosmetics	1,139	89	359	429	142	49	34	8	29	5.9
Household Prep.	3,311	170	1,299	961	330	160	153	55	181	17.1
Bleaches	935	54	308	265	99	51	43	25	110	4.8
Lye	344	11	88	85	44	40	27	8	17	1.8
Other	2,032	125	903	611	163	69	83	22	54	10.5
Solvents	1,020	45	427	299	98	34	43	18	64	5.3
Turpentine	232	10	104	69	16	5	12	2	14	1.2
Kerosene	188	10	93	55	13	7	5	2	3	1.0
Other	608	25	240	175	59	22	26	14	47	3.1
Insecticides	1,231	106	447	340	121	75	70	9	63	6.4
Rodenticides	474	37	164	122	47	28	23	7	26	2.4
Miscellaneous	3,778	260	1,199	1,119	484	223	110	101	82	19.5
Lead	650	7	169	273	122	40	35	3	1	3.4
Other	3,128	253	1,030	846	362	183	275	98	81	16.1
TOTAL	19,357	1,001	5,284	5,992	2,857	1,152	1,091	482	1,498	
Percent	100.0	5.2	27.3	30.9	14.8	6.0	5.6	2.5	7.7	

The figure 650 (lead) includes possible lead poisoning cases

The high incidence of aspirin poisonings is a case in point and clearly illustrates the relation of availability and accessibility to the frequency of poisonings. Because of the low cost of the product especially when bought in large quantity, families succumb to the sales pressure and buy inordinately large amounts of aspirin without relation to need. We would strongly recommend that aspirin, as all other drugs, be taken only on the advice of a physician and that physicians prescribe small quantities of any medication sufficient for the immediate need and when an over-supply is available that it be discarded carefully, preferably in the incinerator, so that it may not be obtained by unsuspecting children who will swallow almost anything they can get hold of.

It may also be said, categorically, that in spite of many claims at the present time, there is no effective safety closure and the utmost care must be employed in keeping all products practically under lock and key and securely away from children.

FATALITIES

Table 3 shows fatalities in individuals under 20 years of age by type of toxic agent. It is noted that in children under 5 years

TABLE 3 — FATALITIES
CHILDREN UNDER 20 YEARS OF AGE
1955 - 1959

Toxic Agent	0-4 Years	5-9 Years	10-19 Years	Total
Lead	68	1	—	69
Methyl Salicylate	4	1	3	8
Aspirin	4	—	2	6
Barbiturates	1	—	3	4
Narcotics	—	—	4	4
Internal Medications	3	—	1	4
Insecticides	2	—	2	4
Rodenticides	2	—	1	3
External Medications	2	—	1	3
Tranquilizers	—	—	2	2
Solvents	2	—	—	2
Methyl Alcohol	1	—	—	1
Detergents	1	—	—	1
Furniture Polish	1	—	—	1
Plastic Bag	1	—	—	1
Miscellaneous	—	2	2	4
TOTAL:	92	4	21	117

of age, the chief cause of death was lead poisoning which, incidentally, accounted for over 70% of all fatalities in this age group. As was stated in other reports, lead poisoning kills more children under 5 than any other illness or disease condition.

Fatalities were also due to methyl alcohol, aspirin, internal medications, external medications, rodenticides and insecticides, methyl alcohol, detergents and furniture polish. Due to the heroic health education efforts, only one death was attributed to plastic bags.

LEAD POISONING

One of the major problems confronting this Center and the New York City Department of Health is the prevention of lead poisoning. Of the 650 children reported to be suffering from lead poisoning between 1955 and 1959, 574 cases were eventually confirmed. Sixty-nine (69) such children, in the 0-5 years of age group, lost their lives from the ravages of this disease, which is totally preventable. Lead poisoning by age, sex and color is shown in Table 4. No statistically significant sex variation is observed. A marked preponderance in the non-white and Puerto Rican is

TABLE 4 — LEAD POISONING CASES 1955 - 1959
Under 30 Years of Age

Race and Sex	Under 2 Yrs.		2 Years		3 Years		4 Years		5 Years		6 Yrs. & Over		T o t a l	
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
White														
Male	14	0	20	2	6	2	5	0	2	0	1	0	47	4
Female	20	3	28	6	6	0	0	0	1	0	1	0	56	9
Non-White														
Male	47	2	44	7	20	3	3	0	3	0	2	0	119	12
Female	29	6	47	6	24	4	5	0	1	0	0	0	106	16
Puerto Rican														
Male	30	3	54	7	27	3	9	0	4	0	3	0	127	13
Female	33	0	46	9	28	4	13	2	3	1	0	0	130	16
T O T A L:	172	14	239	36	111	16	31	2	14	1	7	0	574	69

•Deaths included under Cases

noted and, as was previously pointed out, this variation is not genetically determined but a function of socio-economic condition.

A marked increase in the incidence of lead poisoning has been reported in recent years. The increase, however, is more apparent than real and is believed to be due to a sharpening of case finding methods employed in the Department of Health's direct health services, such as the child health stations, and to an increased awareness on the part of physicians and hospitals.

Recently, a Study Group on Lead Poisoning was organized. This group meets monthly with a view toward determining the best possible approach to a lead poisoning control program. In cooperation with the Bureau of Preventable Diseases and the Bureau of Sanitary Inspections, a comprehensive study is being made in the "lead belt" where samples from various parts of the apartment are tested for lead content and all children under 6 years of age in the household are referred for a urinary coproporphyrin test. All positive reactors (3 plus and above) are referred for a blood lead determination and if the blood lead is 0.06 milligrams per 100 cc of blood and above, such children are referred to a treatment agency for further investigation and possible treatment.

Table 5 shows the incidence of lead poisoning, fatal and nonfatal, by year, since the Poison Control Center was established. The high incidence in 1959 and, incidentally, even more cases were uncovered in 1960, does not mean that more cases are now occurring but, as stated previously, it means that we are now finding more cases in their incipiency and it is at this stage that they are more amenable to treatment. As a matter of fact, this is well

TABLE 5
LEAD POISONING CASES, FATAL AND NON-FATAL
1955 - 1959

Year	Cases	Fatal	Non-Fatal
1955	110	18	92
1956	94	9	85
1957	85	9	76
1958	114	21	93
1959	171	12	159
Total:	574	69	505

documented by the fact that the case fatality rate has been reduced from 16.4% in 1955 to 7.0% in 1960.

It is noteworthy that due to the additional efforts in case finding currently employed, nearly 50 cases of asymptomatic lead poisoning in children under 5 years of age are uncovered annually. It is strongly recommended to practicing physicians and hospital staffs that whenever a child comes to their attention on whom a specific diagnosis could not be made and who is suffering from ill-defined symptoms, that a blood lead determination be made. We believe that such a practice will bring high returns.

May we also suggest that when a positive lead poisoning case is found or when a child with pica is uncovered that the siblings also be subjected to blood lead determination. We find a high index of correlation.

One wonders, since this approach has been so productive in uncovering asymptomatic cases of lead poisoning, whether it would not be just as profitable in studying other non-infectious diseases, such as naphthalene poisoning.

The data presented here is derived chiefly from reports submitted by physicians and hospitals. The educational import of these findings is of inestimable value. It is however limited because of the incompleteness of reports and also because of many events which go unreported. A strong plea is therefore made to all practicing physicians to report all incidents promptly and in every detail to the Poison Control Center so that educational material based on these reports may be disseminated to the profession without much delay and in more complete form.

125 *Worth Street, New York* 13

(*This is the eighth of a series of papers by Dr. Jacobziner*)

Clinical Conference . . .

TREATMENT OF ACUTE POLIO

THE ROOSEVELT HOSPITAL, New York
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EDMUND N. JOYNER, III, M.D., *Chief of Pediatrics, Presiding*

PHILIP MOEN STIMSON, M.D., *Professor Emeritus, Clinical Pediatrics, New York Hospital-Cornell University Medical College, Consultant in Pediatrics, Roosevelt Hospital, Guest*

DR. JOYNER: We are fortunate today in having Dr. Philip Stimson talk to us on the management of polio, with which disease he has had very considerable experience—both in the hospital and as a clinician and teacher.

DR. STIMSON: Today, in discussing polio, there are some interesting highlights which I shall attempt to outline, but with time limited as it is, I obviously cannot cover all aspects of the disease. I would like to start with some statistics. So far this year in the United States, there have been slightly under 3,000 cases, which is far better than any year, for some years past. We will probably have only about 3300 this entire year (1960), which is in tremendous contrast to the 57,000 we had in 1952.

In 1956 the Salk vaccine began to be used fairly generally and we had only 15,000 cases that year. The next year 5,000; about the same for 1958, but in 1959 the rise went up to 8,500. This year, the number of cases took a drop which led many people to think that it is because most children have had the Salk vaccine. But in 1942, long before we had the Salk vaccine, there were only 4,167 cases in the United States and back in 1938, only 1,705. So, a natural wave in the incidence of the disease may have something to do with the decrease during the current year, rather than the vaccine.

As a matter of fact, almost half the population of the United States—people of all ages—have not yet had polio vaccine. Most of these people are over 40. The figures as of last April 1960 showed roughly 20% of little children up to 5 years of age had not had any vaccine; 8% of children 5-19 years had had no vaccine; of adults from 20-39, 45% in this country had had no

vaccine, and over 40, 89% had had no vaccine. So there is a long way to go still in immunizing (as well as Salk vaccine can immunize) the population in the United States. So much for statistics.

Before discussing polio clinically, I want to tell a story for what it is worth. Several years ago, a nurse in one of the best training schools here in New York had her day off on Friday. Thursday she began to feel rotten, but had a date Thursday night and went out, was very active all day Friday, and barely dragged herself back to the hospital Friday night. Saturday morning she was diagnosed as polio and Monday she was dead. Here is a case of polio which was not recognized early and in which the patient kept very active for the first 36 hours or so. In contrast, another nurse from the same hospital was down at Willard Parker Hospital on the Polio Service as an affiliate. She had heard a lecture on the importance of early rest, and one day at luncheon, she said to her room-mate, "I've a headache". Her room-mate said, "We're going to take your temperature"; she had 100.4 by mouth. The room-mate put her to bed. A house officer was called. He thought she had a little stiff neck, and put her in a private room.

I came to the hospital about 4 o'clock and was asked to see her. I asked her if she would sit up and see if she could kiss her knee. She missed it by about 6 inches. Her temperature at the time was 102 by rectum. I asked her if she was inquisitive about the number of cells in her spinal fluid, and she said she was. A lumbar puncture was done and she had approximately 60 cells. This does not prove polio, but *does* prove something wrong with the central nervous system—in her case probably polio. She was kept quietly in bed practically from the very outset of her illness for a period of something over two weeks. Then she was allowed up gradually, and in five weeks she was back on duty, perfectly normal, with no weaknesses of any sort. Of course, this does not prove anything but it illustrates how one patient who kept active died, and another, who was put to bed from the very onset of the disease, developed no weakness.

Now, I shall say something about recognizing the disease and about its early treatment.

There are a lot of other viruses now-a-days that apparently can cause polio-like manifestations, such as the so-called ECHO viruses. And although the polio virus infections are slowly

diminishing in incidence, it is important to recognize polio-like diseases so that they can be treated properly from the start. The early manifestations are those of involvement of the central nervous system and have the characteristic features of fever, headache and stiff neck. During the polio epidemic years, around 1950, mothers were pretty panicky about a child who had fever, and would telephone greatly perturbed. I used to say to them, "please ask him to kiss his knee". "Do what?" "Ask him to kiss his knee" I would repeat. She would say: "how?" I would reply "Any way". And she would say: "yes, he can kiss his knee perfectly well". "Then he has not polio"!

If he has any degree of stiff neck, a child will not be able to kiss his knee; he will miss it by several inches, and in a severe case of polio, an adult cannot come within 25-30 inches of kissing his knee—measuring from the hairline of the forehead to the patella; incidentally that is one of the ways to measure during treatment the progressive relaxing of the back, and in physiotherapy it is a very proud youngster who can get down to 10 inches; so kissing the knees is one sign.

Changes in reflexes are the next important thing, and the easiest ones to watch are the changes in the four quadrants of the abdomen. Early in the disease, they are apt to be a little bit exaggerated and then one quadrant will go out ahead of another, and pretty soon you will find one quadrant increased and one quadrant gone, and two more or less average, but within a few hours of the onset of the disease, the four quadrants of the abdomen in most cases, are all gone.

Changes of the reflexes of the knee jerks, and of the ankle jerks are of some significance. As long as the reflexes are still present, you know you have not much weakness because you have to have intact anterior horn cells to have a reflex arc present.

Another sign which is very common is tremor. Give a child half a glass of water to hold at arm's length, and many will almost spill it because of tremor. This is a suggestive sign, particularly of the possibility of weakness developing in that arm. Tightness of muscles other than in the neck, spine and hamstrings, and transient weaknesses, may appear early if they are looked for. The latter usually are overlooked. With the patient in bed, it is hard to pick up a little weakness, but it is important that this be looked for in great detail before the patient is said to be able to be up and around as a non-paralytic.

Vasomotor disturbances are rather common such as dermographia, sweating behind the neck, and mottling of the skin. Most of the patients show a mixture of irritability and insomnolence. If you leave them alone, they will be a little dopey. If alarmed, they immediately become irritable. They are afraid you are going to move them, and they have learned that being moved hurts, whereas if they lie perfectly still, it does not hurt too much. That is the key to the early treatment of the disease.

As I said at the start of this talk, the thing that is most important in the initial treatment of early polio is absolute rest. On our polio service we had a standing order that any patient with fever must not be wakened for any reason whatsoever, such as rounds, physiotherapy, meals, to have his temperature taken at 6 o'clock in the morning, or for a routine bed-pan. . . Natural sleep is the most important treatment in the early care of acute polio.

When you want to lift a patient, you do not pick him up by the thigh and calf but by the ankle and the knee because the muscles are tender. Hot packs are comforting although not curative. A patient with not too much fever but with sore muscles you can turn on his face, but remember there's an art to turning a polio patient on his face without hurting him. Roll him a little bit toward you and tuck his further arm in under his body, then quickly flip him away from you over on his face. (It is very easy if the arm is out of the way.) Then, with him lying on his face, you can put on flat hot packs—it is not necessary to wrap them around him because most of the tightness and soreness are in the posterior part of the patient, in his neck, back, hamstrings and calves, rather than around the front. Put on the hot packs, change them every ten minutes and leave the hot packs on until he wakes up. Such a treatment is very comforting and relaxing.

Sedation is forbidden in the presence of acute polio because if you put a patient to sleep, and he should develop respiratory difficulty from any one of a number of causes, he may die in his sleep; the respiratory difficulty may require voluntary breathing effort to keep going. So sedatives in the acute stage of polio are definitely contra-indicated. You can give him aspirin to make him more comfortable and you can put hot packs on him. Then the chances are he will go to sleep of his own accord. Many of the patients complain of cramps in the acute stage and small doses of quinine handle this very nicely. Most of the patients are constipated, but cathartics are forbidden. If you give them a

cathartic, it stimulates the intestinal peristalsis, but the constipation is not due to lack of intestinal peristalsis, it is due to inability to relax the sphincter of the anus; the lower rectal muscles do not function and the cathartics are not effective. All you do when you give him cascara is give him a bad stomach-ache. The way to treat constipation in acute polio is with an enema every other day. This is usually sufficient to keep the bowels from becoming impacted.

In order to give every acute polio patient the best possible chance, all wards should display these signs: don't waken . . . don't hurt . . . don't tire!

In any kind of coma, the sense of hearing is the first to return. You may think a patient is comatose, just barely comatose, and you may say something in his presence thinking he does not hear you. After he has come out, he will tell you what you said. So be careful what you say in the presence of a patient you think is comatose, if you do not want him to quote you afterwards! Remember, when going into coma, hearing is the last sense to disappear, and it is the first one to reappear.

Regarding lumbar puncture, when you suspect polio, if you have a patient in a home who has a little stiff neck, a little fever, and cannot kiss his knees by 6 inches and you are wondering what the result of the lumbar puncture is, it is not always wise to send the patient to a hospital to have this done. What if the spinal fluid does show 55-60 cells or more, it merely means there is something a little wrong with the central nervous system, though not necessarily polio. If you are concerned for the best welfare of such a patient, the thing to do is leave him in his own bed at home and watch him. The chances are that the next day he will be better. If he is worse, consider the hospital. But I feel very strongly that many patients with incipient polio are made worse by the fatigue of the trip, the hospital examinations and the nervous excitement.

In consultation at one of the big hospital centers, I once saw a patient on a Monday morning, who had been admitted Friday afternoon. I looked at the chart and eleven different doctors had made complete physical examinations on that patient between Friday afternoon and Monday morning. The patient, an 18-year old girl, complained of being very tired. They had been having trouble making a diagnosis, and it was about the worst way to treat that patient. She should not have been subjected to all that

handling. Have one doctor do an examination on behalf of several doctors and get some idea of the diagnosis, and let no doctor do a thorough muscle examination in acute polio. It is going to change from day to day anyway if there is muscle involvement. It is not necessary to know exactly what muscles are involved at the start.

Placing a patient in the respirator depends first on the type of polio the patient has. Let us discuss first the diagnosis of bulbar polio and start with an illustration: I was called in consultation to one of a neighboring town's good hospitals to see a lovely 21-year old girl. They realized she had polio and she was "gatched up" in bed in an oxygen tent drowning in her own secretions. She could not swallow and she could not cough. She was puddling the saliva in the back of her throat. She was being given oxygen and there she was, sitting up, semi-erect. Of course, the first thing to do was to take her out of oxygen, put her down flat and raise the foot of the bed—turn her over on her side and say "drool, get the saliva out", and pretty soon she did, found she could breathe, and felt better. What happens when you cannot swallow, nor cough, is that you puddle saliva in the back of your throat and cannot get rid of it unless you drool, so it must run out, or must be suctioned out.

So we should try, by all means, to recognize bulbar polio when it is present. We should recognize that it is not primarily a question of lack of oxygen; it is a question of mechanical obstruction between the outside world and your lungs. A lot of saliva is in there that is interfering . . . it is a definite barrier.

Now, bulbar polio is called bulbar because the medulla oblongata or bulb, is the center for the 9th, 10th, 11th and 12th cranial nerves. These are the nerves that control the palate, the pharynx, the tongue and the power of swallowing. There is a good deal of overlapping between the functions of the nerves so it need not be said this patient has a 9th or 10th nerve involvement, but instead, that the patient has weakness of the palate, or weakness of the tongue on one side, or weakness of the muscles when swallowing. In the larynx, the abductors or the adductors of the vocal chords may also be involved in bulbar polio.

Therefore, a first thing to look for is whether the palate is functioning normally and there are four signs of trouble: first, there is a change in the voice. The patient talks as though he had a hot potato in his mouth. Second, is nasal regurgitation. Fluid comes back through the nose—this is apt to be the first thing

that is noticed. The patient takes milk or water and it runs out of the nostril because the function of the soft palate is not too good. Third, the patient loses the gag reflex. One can tickle the palate with a tongue depressor, and the patient does not gag. The uvula is just limp there. Fourth, ask the patient to say "ah". With the average person, the palate goes up in the mid-line. If it is weak on one side, it will rise to the strong side, not go up straight but kick up to one side. Another sign to watch for at the same time, is the lateral walls of the pharynx. When we ask the patient to say "ah", the side walls are apt to move toward the mid-line. When there is pharyngeal paralysis, they stay pretty much fixed in place. Weaknesses of the palate are early signs, and particularly with regurgitation of fluid through the nose, give evidence of bulbar paralysis. If there are any of these symptoms, the patient had better be in a hospital. On the other hand, if when first seen he has already shown signs of improvement, he can be watched carefully at home for a while. I have had mild bulbar home patients. The disease did not develop further and they got well, although each had had definite but slight nasal regurgitation for a day or two. We left them absolutely quiet, and the polio subsided.

The next evidence of weakness you look for is in the tongue. The average person can stick his tongue out and if you ask him to move it from side to side, he can do it perfectly well. When one side of it is weak, and he sticks his tongue out it curves around to the weak side. If the tongue is paralyzed on both sides, it is very much in the way when he is lying on his back, and it may fall back into his pharynx and be an actual mechanical obstruction to breathing. Those patients ought not to be allowed to lie on their backs.

When you give the patient something by mouth and he chokes—he cannot swallow it, and there is puddling in his throat—it is very obvious that there is weakness in swallowing.

The coughing mechanism is a complex one. When a patient coughs, he has to do three things: close the vocal chords to get something to cough against, fix the diaphragm, and contract the abdominal muscles rather sharply. Fixing the diaphragm helps the contraction of the abdominal muscles. That compresses the chest and air going out runs up against a closed larynx, which gives increased pressure and a cough is developed. A sneeze is a similar mechanism, except it is not against the larynx. It is

against the soft palate. So, a patient may be unable to cough if there is paralysis of the vocal chords, or if there is a paralysis of the diaphragm, or weakness of the abdominal walls. When you cannot cough, you cannot get mucus out of your trachea. The liquid mucus is another mechanical obstruction to breathing and the patient may drown if he cannot cough. It is another reason for postural drainage. Thus, the mechanisms of inability to breathe in polio are very complex. I have just hinted at the bulbar aspects. They may be due entirely to bulbar difficulties. The patient's muscles of breathing, his intercostals, his diaphragm and the telephone central that telephones to those muscles may be all right, but he has this mechanical barrier in the pharynx between the outside world and his lungs, past which he cannot get oxygen. There is mucus in there, or a tongue that is in the way or the patient cannot cough. Put such patients in a respirator and they will die if you do not first do a tracheotomy. All the mucus in their throats just sucks down into their lungs and they die. It took several years to find this out when respirators first came out. Bulbar cases were put in respirators and they died, so pretty soon, we decided we might as well let them stay out. Then it was decided to do tracheotomies and have these patients breathe through their necks instead of through the larynx.

If there are other manifestations of bulbar difficulties, such as palatal weakness or pharyngeal weakness, we are also apt to find involvement of the breathing central and that is shown by irregular breathing. If we ask the patient to take a big breath, he can do so if the intercostals and diaphragm are all right, but then, if you leave him alone a little bit, the breathing is irregular and apt to be shallow, and he cannot sleep.

The other center in the medulla, which is important for life, controls the size of the blood vessels and is called the vasomotor center. When it fails, the patient goes into shock, the blood pressure goes down, and the patient dies. That is why bulbar polio is so serious and why there is such a high mortality.

We can treat the patient who has puddling in his throat; we can treat the patient who has a laryngeal obstruction; we can treat him if he has a palatal paralysis, but you cannot do anything for a failure of the vasomotor center. You can whip it up for a little while with various drugs, but it goes down and out, and the patient dies. With respiratory center involvement, you can put a patient in a respirator even though he does not coordinate

with the machine very well. It is very difficult, with a respirator, to save a patient who has involvement of the vasomotor center.

There is another kind of respiratory difficulty which has to be sharply differentiated. The respiratory difficulty we speak of here, is due to weakness of the muscles with which one breathes—primarily the intercostals and the diaphragm. This can be measured with spirometers to find out how bad it is. The average adult normally breathes 4,000 cc in each breath; the polio patient goes to where he can breathe only 1200 or 1500 cc in a breath . . . well, that is about the borderline. He had better go into a respirator if he is down that far, if only to give him some sleep. But after he has been in a respirator, if you are going to wean him from it, you will often find he can get along pretty well on 700-800 cc for a while.

All of this you cannot learn in 15 minutes. I am just showing you the complexity of breathing difficulties in polio. It takes a great deal of experience. I have heard one polio expert say that if he were in a hospital where there was a respirator but nobody knew how to use it, he would rather not be put in it, unless someone knew how and when to use it. You do not place a patient in a respirator just because he has some breathing difficulty. The respirator has to be used wisely and skillfully; if not, you can kill the patient. Now, do you want me to go on with breathing difficulties? There may be questions . . .

DR. JOYNER: I can tell you what you might do: summarize your criteria for placing a patient in a respirator.

DR. STIMSON: Well, in the first place, if it is a pure spinal case with breathing difficulty, you put him into a respirator before he gets too tired, and that usually means when he is breathing 1200-1300 cc per breath; you may be able to take him out for an hour a day right after the first day. But at least, he will have a decent night's sleep. We are putting spinal cases in a lot quicker than we used to.

With a combination bulbo-spinal case, one should always do a tracheotomy before you put him in. You cannot do it very easily when he is in the respirator because the collar is around his neck and in the way. We have done it, but it is awkward. A pure bulbar case you do not put in. You put him on his side. There is a trick in doing that, which nurses as well as doctors ought to know. Don't raise the foot of the bed very high, about 15 degrees is enough. You turn the patient well over on his side, with his

head on a small pillow to be in line with his spine, with the lower arm on the bed, flexed at the elbow, and the upper arm across a big bulky pillow. It is quite comfortable if the upper arm is on a pillow and not lying across his chest and interfering with his breathing. You also take a big pillow and ram it up against his back so he does not roll. You put a pillow between his flexed knees so that he is comfortable, and you have just enough under his head to keep it in line with the spine and then you put a kidney basin or a handful of gauze where he can drool into it, unless you have somebody constantly on the job with an aspirator.

An advantage of this position is that the veins in the forearm or elbow on the bed and in the back of the hand or wrist on the pillow are available for intravenous feedings. The patient can keep that position very comfortably for 2-3 hours, and then you can put him over on the other side. Can you visualize that position? It is really quite important for the treatment of bulbar polio.

DR. JOYNER: Are there any questions?

VOICE: What happens if they are unable to breathe with bulbar involvement and the respiratory center is out?

DR. STIMSON: Do a tracheotomy and make the machine breathe for them. You must not have them suck all that secretion down into their chest.

VOICE: You mentioned the fact that you kept these patients isolated until the temperature came back to normal. You also mentioned the fact that the virus could be picked up in the stool until 17 weeks later.

DR. STIMSON: The patient is in contact with nurses, doctors, the family and everybody else. Most of them are infected. As far as the stool is concerned, what is the sense of closing one window in a house and leaving all the others wide open. The virus is present in the pharynx only during 2-3 days of fever, but it should be gone by the end of the fever. With regard to isolation, I think the New York City Health regulation is "as long as there is fever and one day more", to be on the safe side.

DR. LANDON: You were talking when I came in, about oral vaccine. What is the feeling now about that versus Salk?

DR. STIMSON: Statistics vary. One of the organizations, I think it was the National Foundation, said the oral vaccine gave at least 95% protection in antibodies. Others have not agreed. When

the Salk vaccine came out, we heard that three shots protected 75%; four shots perhaps 90%, and those are the figures usually given. But at a meeting of the Advisory Committee of the Board of Health the other day, much lower percentages were reported. The Board of Health at its health stations is giving quadrigen which is the polio vaccine with DPT. At the New York Hospital, Dr. Eichenwald did a number of studies on the efficacy of the polio element and found certain preparations in which its immunizing property in quadrigen was unsatisfactory. Also, there are a few preparations of polio vaccine alone which were unsatisfactory, so, at New York Hospital they are not giving quadrigen. They give polio and DPT in two syringes at the same time, and I have been doing that in my own office. I spoke about this at a meeting the other day and one of the doctors said, "oh, it is perfectly all right to give the quadrigen; the only thing is it lowers the effect of the whooping-cough element a little bit to have it altogether". What are you going to do? Well, I'm going to go on giving them separately. This morning I gave a couple of youngsters polio in one buttock and DPT in the other, and I am going to go on doing that until I get some better evidence.

DR. JOYNER: The advantage of the quadrigen is that even if it is not as well known as the separate vaccines, you give one less injection. Now, it is perfectly true that triple vaccine stings, and if you give the polio which does not sting while the triple one is stinging, they don't even feel it. They don't change expression. Do you give a fourth shot?

DR. STIMSON: I give a fourth and if people ask for a fifth, I let them have it a year later. Why not? It doesn't do any harm.

VOICE: What I want to know is whether or not you start at 2 months, as is now recommended by the Academy of Pediatrics, and if so, do you give a third polio 7 months later, and then a so-called booster?

DR. STIMSON: It depends on the time of year. If it is Spring, when I get a baby for the first time, I inject him three months in a row so as to get them in before summer. Right now in January I give two, and then I wait until late Spring for the third.

VOICE: Do you start at 2 months?

DR. STIMSON: I give my first polio shot at 2 months. As you know, there was a meeting at the Academy last year on this. I think somebody started at six months.

VOICE: But it isn't as good?

DR. STIMSON: No, I think 2 months is better.

VOICE: Six months is what they used to say. Most children had some antibody response as early as 1-2 months, but the great proportion did not get it until six months.

DR. STIMSON: What I have been doing is polio at 2 months, polio and a triple at 3 months, triple at 4, triple at 5. Then the third polio depends on what the relation is to the next summer, and then a year later give a booster of each. For the first vaccination, I wait until they are six months old. One of the medical students at Cornell at my suggestion looked up the literature on vaccination at birth, and found it was compulsory in France and in Poland. They do it the day of birth. He also found that vaccination done at that time "doesn't stick", that is, if you vaccinate the child again at 1 year of age, you often get another take, and therefore there is no point in vaccinating earlier than six months unless there is actual exposure.

DR. JOYNER: I know we have all enjoyed Dr. Stimson's talk. I think the one thing most of us got from it and which I know Dr. Stimson would like to have you carry away is that the less you do for an acute spinal polio the better. There is no cure for it except time and rest. Whatever cure and whatever help we can give is to help nature, and then later on, get busy in the physiotherapy and other methods. But during the acute stage, if there is no bulbar, and no respiratory involvements, you cannot do anything more in a hospital than you can at home. The more you leave them alone and make them comfortable, the better they are going to be. Of that I am absolutely sure. Dr. Stimson taught me that many years ago, and I am very thankful for it. There is no drug, there is nothing except nature, and you cannot help nature by putting the patients in an ambulance, taking them 75 miles or through 8-10 examinations as Dr. Stimson said, to learn which muscles are involved. It simply tires them and involves more muscles, so leave them alone. Thank you very much, Dr. Stimson.

The Cutaneous Evaluation of Verel Fabric Diaper Liners

Raymond R. Suskind, M.D.,* Gyula Haniszko, M.D.,*
William Cohen, M.D.*
Ohio

One of the most common eruptions encountered in infants and young children is dermatitis in the diaper area. The inflammatory reaction in many instances is initiated by prolonged contact with the wet diaper and its contents. The continuous wetting of the skin produces maceration, damages the epidermal barrier and makes the skin more vulnerable to penetration by potential irritants and microorganisms. The wet, warm diaper is also a good growth environment for urea splitting organisms (Bacterium ammoniagenes) which form ammonia and which may provoke irritation. The irritation so initiated may then be complicated by infection with pyogenic organisms or in other instances with fungi such as Candida albicans.

Since prolonged wetting and the maceration it produces, is a primary factor, any technique for maintaining a relatively dry skin would appear to be potentially useful in preventing this cutaneous problem.

We have had under study, a new knitted fabric made of Verel† a synthetic modacrylic fibre. This material in suitable sizes can be used as a diaper liner. The unique low moisture retention properties of the fibre and the porousness of the fabric allows the urine to pass through the liner to be absorbed by the outer cotton diaper, thus keeping the skin surface adjacent to the liner dry, following urination.

Two series of experiments were conducted. Although they were originally designed to appraise the possible irritant or sensitizing effects of the fabric on human skin, the repeated observations provided significant information regarding effectiveness as well.

* From the Kettering Laboratory in the Department of Preventive Medicine and Industrial Health and the Department of Dermatology, College of Medicine, University of Cincinnati.

† Verel is a product of the Eastman Chemical Products, Inc., and the diaper liner material was supplied by Eastman Chemical Products through the courtesy of Dr. David W. Fassett, Director, Laboratory of Industrial Medicine, Eastman Kodak Company.

The studies consisted of: 1) a repetitive patch test appraisal in adults. 2) a three-month usage test in infants and children.

REPETITIVE PATCH TEST APPRAISAL

1. *Procedure*: The materials employed in this test consisted of #1-a knitted fabric prepared from Verel fibre, #2-a commercially available birdseye cotton diaper which is extensively used in the U. S. (Control).

Squares of fabrics #1 and #2, measuring 4x4 inches were applied upon the skin of the back of each test subject and retained in place with adhesive tape 2 inches in width. The patches were kept in place for 4 days during the initial period of contact, after which they were removed and within 30 minutes, observations were made for cutaneous reactions. Fourteen days after the initiation of the first application, the test materials were applied upon the skin of the back of the same subjects at approximately the same sites and allowed to remain in place for 48 hours. Following the removal of the fabric patches, a period of 30 minutes was allowed to elapse and observations were then made for cutaneous reactions. The purpose of the initial period of contact was to provide information regarding the primary irritant capacity of the fabrics and to afford a means, if possible, of sensitizing the skin. The reapplication or challenging procedure was an attempt to determine if any of the subjects exposed previously had become sensitized allergically to the components of the fabrics.

The criteria established in advance for grading skin reactions in accordance with previous experience were as follows:

1+ mild but definite erythema; 2+ moderate to severe erythema or erythema and edema; 3+ erythema and vesiculation or vesiculation alone. In the primary exposure series, 199 persons were tested to the two materials. Of these, 191 completed the second series of tests two weeks later. All of the 199 subjects were white persons with normal skin.

2. *Observations*: No skin reactions which could be interpreted as due to either primary irritation or allergic sensitization, were observed. Although a significant portion of this study was conducted during hot summer weather, no maceration or sweat retention phenomenon was observed following application of the test or control fabrics. The results of the patch tests are presented in Table I.

TABLE I

Patch Test Observations—First Series

(96 Hour Application)

Fabric	Number of Subjects	Negative Reactions	Positive Reactions 1+	2+	3+	Total Number Positive Reactions	Per cent Positive Reactions
#1	199	199	0	0	0	0	—
#2	199	199	0	0	0	0	—

Patch Test Observations—Second Series

(48 Hour Application)

Fabric	Number of Subjects	Negative Reactions	Positive Reactions 1+	2+	3+	Total Number Positive Reactions	Per cent Positive Reactions
#1	191	191	0	0	0	0	—
#2	191	191	0	0	0	0	—

USAGE TEST

1. *Procedure*: The materials employed in this test consisted of the following: a Verel fabric diaper liner measuring 11 x 17 inches, that constituted the inner portion of the baby diaper complex; a standard birdseye cotton diaper that constituted the outer portion of the diaper complex.

Each mother was supplied with 5 dozen cotton diapers and 5 dozen diaper liners. The diaper liner and cotton diaper were used continuously on all the subjects for a period of three months. Cutaneous examinations of the areas exposed to the diaper complex were made prior to the initiation of the test and at monthly intervals for a total of 4 to 5 observations. The mothers were instructed carefully in the proper use of the liners and their laundering at the initial meeting of the test groups. Instructions were given to wash in water not over 140° F and if mechanical dryers were used, not to exceed this temperature. In all instances, the diapers and liners were laundered in either automatic or semiautomatic washing machines with commercial laundry soaps or synthetic detergents.

The mothers were asked to observe the skin of the diaper area during the course of use and watch for any abnormal changes in the skin. In the case of children who were inclined to develop irri-

tation of the skin in the diaper area, mothers were requested to observe for improvement or aggravation of the eruption. At each subsequent meeting the mothers were questioned regarding the usefulness and laundering qualities of the Verel liner as compared with standard cotton diapers. Finally, a printed questionnaire was distributed to the mothers at the third or fourth examination session requesting specific information.

The primary information regarding the possible effects of the diaper liner fabric was obtained by periodic clinical examination. Additional valuable information was obtained from the day-to-day observations by the mothers during the three-month test period.

2. *Subjects*: One hundred and five, (105) babies in good health, were exposed to the continuous use of this double-layer diaper containing the Verel liner without regard for the condition of the skin in the diaper area. Their initial age range was from 3 weeks to 26 months old. There were 62 males and 43 females among them. At the beginning of the test, 71.4% of the subjects were free of any eruption in the diaper area, the remainder demonstrated eruptions ranging from mild redness to an erythematous papular rash in this area. None of the eruptions were severe enough to require medical treatment.

3. *Observations*: During the 3 month period, no adverse reactions which might be attributable to the diaper liner were noted among the 105 subjects. Thirty subjects were observed to have some eruption in the diaper area prior to the use of the liner. In 22 of these, the erythema subsided completely before the end of the test, (Table II). Eighteen cleared before the second examination. One of these was a child with atopic dermatitis who, previous to the test, had a long standing eruption in the diaper area. In 8 of the 30 subjects cited, some mild erythema was present at the final examination. The redness in 4 of the 8 children cleared during the 3 month period but was again noted during the last observation. Of these, one was directly related to wearing plastic or rubber pants; one was attributable to recurrent diarrhea, and there were two siblings who had a mild rash throughout the test.

It was observed that, in most instances, when the liner was employed the skin of the children was dry at the first diaper change in the morning, which was usually 8 to 10 hours following the last evening change. The mothers were of the opinion that those children who had the tendency to develop diaper rashes occasionally or continuously had such reactions very infrequently during the test

period in which the Verel liner was used. Maceration of the skin was never observed. It was not necessary to employ a dusting powder or oil in the diaper area so long as the liner was employed. The diaper liner did not prevent irritation from stool contents. In some instances in which the mothers allowed the children to remain in contact with feces for prolonged periods, moderate irritation developed. This cleared without difficulty when the children were changed more promptly after defecation. During transient bouts of diarrhea of one cause or another some of the children developed diaper rashes despite use of the liner.

TABLE II

Cutaneous Response to Verel Diaper Liner

Total Subjects	Normal Skin		Subjects with Eruption in Diaper Area Prior to Test	
105	75	71.4 %	30	28.6 %

Number of Subjects Cleared	% Cleared
22	73.3

CONCLUSION

From the patch test observations and a trial usage appraisal, one can conclude that the Verel diaper liner fabric will neither irritate nor allergically sensitize human skin. It was further demonstrated that the unique physical properties of the fabric provide an effective and useful means of keeping the skin of diapered children dry and in doing so can limit the incidence of diaper dermatitis in which continuous wetting and maceration are significant factors. Those children with diaper area reactions provoked by factors other than wetting alone, e.g., bacterial and fungal infection may also benefit from use of the Verel liner since maintaining a dry skin surface is likely to discourage the growth of microorganisms.

Editorial Note: Since diaper dermatitis is a perennial concern of both pediatricians and mothers, it would appear from the foregoing observations that this mechanical means of handling this stubborn problem warrants further study.

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1. The Composition of Milks, Publication 254, National Academy of Sciences and National Research Council, Revised 1953. 2. Brown, G.W.; Tuholski, J.M.; Sauer, L.W.; Minsk, L.D., and Rosenstern, I.: J. Pediat. 56:391 (Mar.) 1960.

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